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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/693,480	10/23/2003	Silviu Itescu	0575/66602-B/IPW/BJA	2572
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EXAMINER BUNNER, BRIDGET E				
ART UNIT		PAPER NUMBER		
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary

Application No.

10/693,480

Applicant(s)

ITESCU, SILVIU

Examiner

Bridget E. Bunner

Art Unit

1647

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --
Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☒ Responsive to communication(s) filed on 18 October 2010.
- 2a) ☒ This action is **FINAL**. 2b) ☐ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) ☒ Claim(s) 35, 37, 43, 46, 47, 49-51 and 57 is/are pending in the application.
- 4a) Of the above claim(s) _____ is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☒ Claim(s) 35, 37, 43, 46, 47, 49-51 and 57 is/are rejected.
- 7) ☐ Claim(s) _____ is/are objected to.
- 8) ☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☒ The drawing(s) filed on 01 February 2008 and 23 October 2003 is/are: a) ☒ accepted or b) ☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some * c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
 2. ☐ Certified copies of the priority documents have been received in Application No. _____.
 3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).
- * See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

- 1) ☒ Notice of References Cited (PTO-892)
- 2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
- 3) ☒ Information Disclosure Statement(s) (PTO/SB-08)
Paper No(s)/Mail Date 10/18/10; 3/22/10
- 4) ☐ Interview Summary (PTO-813)
Paper No(s)/Mail Date _____
- 5) ☐ Notice of Informal Patent Application
- 6) ☐ Other: _____

DETAILED ACTION

Status of Application, Amendments and/or Claims

Claims 35, 37, 43, 46, 47, 49-51 and 57 are under consideration in the instant application.

Double Patenting

The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. A nonstatutory obviousness-type double patenting rejection is appropriate where the conflicting claims are not identical, but at least one examined application claim is not patentably distinct from the reference claim(s) because the examined application claim is either anticipated by, or would have been obvious over, the reference claim(s). See, e.g., *In re Berg*, 140 F.3d 1428, 46 USPQ2d 1226 (Fed. Cir. 1998); *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970); and *In re Thorington*, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) or 1.321(d) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent either is shown to be commonly owned with this application, or claims an invention made as a result of activities undertaken within the scope of a joint research agreement.

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

1. Claims 35, 37, 43, 46-47, 49, 50, 51 and 57 are rejected on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 1-6 of U.S. Patent No. 7,662,392. Although the conflicting claims are not identical, they are not patentably distinct from each other because both sets of claims are directed to a method of treating a disorder of the heart comprising administering stromal-derived factor-1. The basis for this rejection is set forth at pages 2-3 of the previous Office Action of 18 September 2009, the Office Action of 03 February 2009, page 4 of the Office Action of 09 May 2008 and pages 6-7 of the Office Action of 08 August 2007. It is noted that in the prior actions, this was a provisional obviousness-type double patenting rejection because the conflicting claims in copending Application No. 11/234,879 had not been patented. The Examiner indicated in the Office Action of 18 September 2009 that a Notice of Allowance was mailed in the '879 application. The '879 has now been issued as U.S. Patent No. 7,662,392.

At page 2 of the Response of 22 March 2010, Applicant argues that the '392 patent discloses a method of increasing trafficking of bone marrow-derived endothelial progenitor cells to ischemic myocardium in a subject comprising administering to the subject an amount of SDF-1 effective to attract bone marrow-derived endothelial progenitor cells to the ischemic myocardium. Applicant asserts that the invention currently claimed describes a method of treating a subject suffering from a disorder of a heart tissue involving the loss or apoptosis of cardiomyocytes which comprises intramyocardially or intracoronarily administering to the subject an amount of an agent comprising a human SDF-1 effective to induce regeneration of endogenous cardiomyocytes. Applicant states that the different cell types and the different

mechanisms of action recited in the present claims are not obvious over the claims of Patent No. 7,662,392.

Applicant's arguments have been fully considered but are not found to be persuasive. Claim 35 of the instant application recites intramyocardial or intracoronary administration of SDF-1 α or SDF-1 β to a subject suffering from a disorder of heart tissue involving loss or apoptosis of cardiomyocytes. Claim 1 of the '392 patent recites administering SDF-1 to a subject effective to attract bone marrow-derived endothelial progenitor cells to the ischemic myocardium so as to thereby increase trafficking of bone marrow-derived endothelial progenitor cells to the ischemic myocardium, and wherein the SDF-1 is administered to the subject by injection into the heart muscle. Claims 35, 46, and 47 of the instant application and claims 2-3 of the '392 patent recite that the SDF-1 is SDF-1 α or SDF-1 β . Claim 49 of the instant application recites that the disorder of the heart comprises myocardial infarction, congestive heart failure, chronic ischemia, ischemic disease, diabetic heart disease or cardiomyopathy. Claim 5 of the '392 patent recites that the subject has suffered or is suffering from myocardial infarction, chronic heart failure, ischemic heart disease, coronary artery disease, diabetic heart disease, hemorrhagic stroke, thrombotic stroke, or other diseases in which the myocardium is rendered ischemic.

Hence, both the claims of the instant application and the claims of the '392 patent recite administration of SDF-1 to the same subject population and to the same tissue. A compound and all of its properties are inseparable; they are one and the same thing and simply stating a new property of SDF-1 does not render the claimed method of the instant application unobvious over the claims of the '392 patent (see *In re Papesch*, CCPA 137 USPQ 43; *In re Swinehart* and

Sfiligoi, 169 USPQ 226 (CCPA 1971); In re May, 574 F.2d 1082, 1090, 197 USPQ 601, 607 (CCPA 1978)).

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

2. Claims 35, 37, 43, 46, 57 are rejected under 35 U.S.C. 103(a) as being unpatentable over Petersen, BE (US 2002/0094327; priority to 05 November 2000) in view of Hung et al. (US 2003/0171294; priority to 13 August 1999). The basis of the rejection is set forth at pages 3-12 of the previous Office Action of 18 September 2009 and pages 3-7 of the Office Action of 03 February 2009.

Applicant's arguments (22 March 2010), as they pertain to the previous rejection of record have been fully considered but are not deemed to be persuasive for the following reasons.

(i) At page 4 of the Response, Applicant indicates that claim 35 recites a method of treating a subject suffering from a disorder of a heart tissue involving the loss or apoptosis of cardiomyocytes which comprises intramyocardially or intracoronarily administering to the subject an amount of an agent comprising a human SDF-1 effective to induce regeneration of endogenous cardiomyocytes and thereby treat the disorder of the heart tissue involving loss or apoptosis of cardiomyocytes in the subject. Applicant submits that the combination of Petersen and Hung et al. does not teach such a method. Applicant argues that Petersen provides a laundry list of tissues to which SDF-1 α could theoretically be administered in order to effect trafficking

of a pluripotent stem cell to the tissue from another site in the subject. Applicant points out, that in contrast, the method as currently claimed induces regeneration of endogenous cardiomyocytes in a specific tissue. Applicant contends that there is no teaching of such a method in the combination of Petersen and Hung et al. Applicant argues that the discussion in Petersen of repopulating a damaged tissue with pluripotent stem cells is not a disclosure of endogenous cells, less so endogenous cardiomyocytes.

Applicant's arguments have been fully considered but are not found to be persuasive. Petersen teaches that a proposed alternative method for treating organ failure is organ regeneration wherein damaged cells of a failing organ are replaced with new, undamaged cells (page 1, [0004]). Petersen discloses that the invention relates to a method for selectively directing migration of pluripotent stem cells to a target tissue within a subject by modulating the level of SDF-1 α protein in the target tissue (page 1, [0006]). Petersen continues to state that by "increasing the number of pluripotent stem cells that traffic to the target tissue, the rate of tissue repair can be increased because there will be a greater number of pluripotent stem cells in the target tissue that can differentiate into cells which can repopulate and partially or wholly restore the normal function of the damaged tissue" (page 1, [0006]). Petersen also teaches that the heart is one of the target tissues within a mammalian subject in which SDF-1 α is administered (page 8, column 2, [0063]). Thus, the teachings of Petersen do not teach away from the instant claims because administration of SDF-1 α to a target tissue (such as the heart) increases the number of pluripotent stem cells (that are already endogenous to the subject) that traffic to the tissue and differentiate into endogenous cells of that damaged tissue.

Since Petersen teaches the administration of SDF-1 to the same subject population and to the same tissue as recited in the claims, the regeneration of endogenous cardiomyocytes must have been inherently occurring in the prior art. The disclosure of Petersen (in combination with Hung et al.) meets the terms of the claimed method because SDF-1 α inherently possesses endogenous cardiomyocyte regeneration activity, absent evidence to the contrary (In re Best, 562 F.2d 1252, 1255, 195 USPQ 430, 433 (CCPA 1977)). A compound and all of its properties are inseparable; they are one and the same thing and simply stating a new property of SDF-1 α does not render the claimed method of the instant application free of the art (see In re Papesch, CCPA 137 USPQ 43; In re Swinehart and Sfiligoj, 169 USPQ 226 (CCPA 1971); In re May, 574 F.2d 1082, 1090, 197 USPQ 601, 607 (CCPA 1978)). Furthermore, inherent anticipation does not require that one of ordinary skill in the art recognize an inherent feature in a prior art disclosure (Schering Corp. v. Geneva Pharmaceuticals Inc., 67 USPQ2d 1664 (CAFC 2003); Toro Co. v. Deere & Co., 355 F.3d 1313, 1320, 69 USPQ2d 1584, 1590 (Fed. Cir. 2004)).

(ii) At the middle of page 5 of the Response of 22 March 2010, Applicant contends that there is nothing in Hung et al. to suggest administration of SDF-1 to the heart intramyocardially or intracoronarily. Applicant points out that Hung et al. describes two animal models of coronary heart disease, a hibernating myocardium model and an ameroid model. Applicant argues that Hung et al. states that hibernating tissue is non-contracting muscle tissue, but is capable of contracting, should it be adequately supplied with blood. Applicant submits that Hung distinguishes dead or diseased heart tissue from hibernating tissue. Applicant asserts that the instantly claimed invention, being directed to a disorder of a heart tissue involving loss or

apoptosis of cardiomyocytes is distinguished from the hibernating model. Applicant states that the data in Hung et al. showing administration of a FGF polypeptide intramyocardially to the hibernating model is not relevant to subjects "suffering from a disorder of a heart tissue involving loss or apoptosis of cardiomyocytes".

Applicant's arguments have been fully considered but are not found to be persuasive. Hung et al. teach "the hibernating cardiac model is a particularly relevant model of coronary artery disease" (page 6, [0038]). Hung et al. continue to disclose that hibernating tissue is non-contracting muscle tissue, but is capable of contracting, should it be adequately supplied with blood while healthy heart tissue is identified by strong electrical signals in combination with strong displacement (page 6, [0038]). Hung et al. state that "[i]schemic, or hibernating or stunned heart tissue is identified by strong electrical signals in combination with impaired displacement" (page 6, [0038]). Hung et al. teach that "[t]he diagnosis of hibernating tissue is critical because it is widely believed that once the occlusion is eliminated, there is an immediate return of normal function...Thus, the hibernating model of the myocardium is similar to what occurs in a human patient having coronary artery disease (CAD) and/or chronic angina wherein one or more coronary arteries are partially occluded" (page 6, [0038]). The hibernating model is clearly not representative of "healthy heart tissue" and the claims of the instant application only recite "a disorder of a heart tissue".

Furthermore, Applicant is reminded that the primary reference of the instant rejection, Petersen, teaches the local administration of SDF-1 α to damaged tissues, including the heart. Although Petersen does not specifically disclose intramyocardial or intracoronary administration of SDF-1 α , Hung et al. teaches that it is routine to administer such factors, such as angiogenic

factors, via intramyocardial and intracoronary administration to the heart (see page 2, paragraphs 9-10; page 4, paragraph 22; page 5, paragraph 34; figure 8; page 22, [0160]; pages 23-24, Examples 7-8).

(iii) At page 6 of the Response of 22 March 2010, Applicant argues that administration of the polypeptide FGF to the ameroid model provided no benefit over placebo (Figures 7-9).

Applicant asserts that Hung et al. shows that administration of FGF to a subject having a diseased heart provided no beneficial effect. Applicant contends that in view of this data presented in Hung et al., a person of ordinary skill in the art would have no motivation to treat a subject suffering from a disorder of a heart tissue involving loss or apoptosis of cardiomyocytes which comprises intramyocardially or intracoronarily administering to the subject an amount of an agent comprising a human SDF-1 because Hung et al. teaches away from intramyocardial or intracoronarial injection of a polypeptide to treat a subject suffering from a disorder of a heart tissue involving loss or apoptosis of cardiomyocytes.

Applicant's arguments have been fully considered but are not found to be persuasive. Contrary to Applicant's assertions, it is clear from Figures 7-9, that the superior and significant dosage of FGF-2 is "mid" (0.6 µg/kg). This "mid" dose resulted in a greater increase in normalized perfusion (particularly at 3 months post-treatment) and a greater vascular density. Regardless of the specific results of the experiments, Hung et al. successfully administer the growth factor, FGF-2, to myocardium. Hung et al. does not teach away from intramyocardial or intracoronarial injection of a polypeptide since Hung et al. teaches that factors can be intramyocardially administered to a subject. Hung et al. also teaches that it is routine to

administer such factors, such as angiogenic factors, via intramyocardial and intracoronary administration to the heart and reviews many references echoing the same (see page 2, paragraphs 9-10; page 4, paragraph 22; page 5, paragraph 34; figure 8; page 22, [0160]; pages 23-24, Examples 7-8).

It would have been obvious to the person of ordinary skill in the art at the time the invention was made to modify the method of administering of SDF-1 α to heart tissue as taught by Peterson by utilizing intramyocardial or intracoronary administration as taught by Hung et al. The person of ordinary skill in the art would have been motivated to make that modification in order to localize cell migration/differentiation and tissue repair (see for example, Hung et al. page 1, [0007]; Peterson, page 1 [0004, 0006]). The person of ordinary skill in the art reasonably would have expected success because similar proteins and agents were already being intramyocardially and intracoronarily administered to the heart at the time the invention was made (see Hung et al.). Therefore, the claimed invention as a whole was clearly *prima facie* obvious over the prior art.

3. Claim 47 is rejected under 35 U.S.C. 103(a) as being unpatentable over Peterson, BE (US 2002/0094327; priority to 05 November 2000 and Hung et al. (US 2003/0171294; priority to 13 August 1999) as applied to claims 35, 37, 43, 46, 57 above, and further in view of Rempel et al. (Clin Can Res 6: 102-111, 2000). The basis for this rejection is set forth at pages 7-8 of the previous Office Action of 18 September 2009 and at pages 12-14 of the Office Action of 03 February 2009.

At page 3 of the Response of 18 October 2010, Applicant argues that Petersen, Hung et al. and Rempel et al. do not render obvious the method of independent claim 35 from which claim 47 depends. At pages 3-5 of the Response, Applicant reiterates arguments against Petersen and Hung et al., as presented in sections 2(i)-(iii), above. At page 5 of the Response, Applicant contends that even if one used the disclosure of Rempel et al. to provide SDF-1 β , it would not have been obvious to one skilled in the art at the time the invention was made to use SDF-1 α in the claimed method which is not obvious over Petersen when combined with Hung et al. and Rempel et al.

Applicant's arguments have been fully considered but are not found to be persuasive. The Examiner's response to Applicant's arguments against Petersen and Hung et al. are presented in sections 2(i)-(iii), above. Additionally, although Petersen teaches the administration of SDF-1 α to heart tissue utilizes SDF-1 α , Rempel et al. teaches that the SDF-1 gene encodes two isoforms, SDF-1 α and SDF-1 β , that arise from alternative splicing (page 102, column 2, last paragraph). Both isoforms interact with the same seven-transmembrane G protein-coupled receptor, CXCR4 (page 103, column 1, 1st full paragraph). Hence, SDF-1 α and SDF-1 β have the same function. Rempel et al. also disclose SDF1-deficient mice exhibit hematopoietic, cardiac, and cerebellar defects (page 103, column 1, bottom of 1st full paragraph). Therefore, it would have been obvious to the person of ordinary skill in the art at the time the invention was made to modify the method of intramyocardially or intracoronarily administering SDF-1 α to heart tissue as taught by Petersen and Hung et al. by substituting SDF-1 α with SDF-1 β as taught by Rempel et al. Since Rempel et al. teach that SDF-1 α and SDF-1 β are isoforms encoded from the SDF-1 gene and that SDF-1 β only contains four additional amino acids as compared to SDF-

1 α , one skilled in the art would have been motivated to substitute the utilization of SDF-1 α for the SDF-1 β to achieve the predictable result of treating a subject suffering from damaged heart tissue. Furthermore, MPEP 2144.09(I) states that a prima facie case of obviousness may be made when chemical compounds have very close structural similarities and similar utilities. "An obviousness rejection based on similarity in chemical structure and function entails the motivation of one skilled in the art to make a claimed compound, in the expectation that compounds similar in structure will have similar properties." In re Payne, 606 F.2d 303, 313, 203 USPQ 245, 254 (CCPA 1979); In re Papesch, 315 F.2d 381, 137 USPQ 43 (CCPA 1963); In re Dillon, 919 F.2d 688, 16 USPQ2d 1897 (Fed. Cir. 1991).

4. Claims 49-51 are rejected under 35 U.S.C. 103(a) as being unpatentable over Peterson, BE (US 2002/0094327; priority to 05 November 2000) and Hung et al. (US 2003/0171294; priority to 13 August 1999) as applied to claims 35, 37, 43, 46, 57 above, and further in view of Isner et al. (WO 99/45775; 16 September 1999). The basis for this rejection is set forth at pages 14-15 of the previous Office Action of 18 September 2010.

At page 6 of the Response of 18 October 2010, Applicant argues that independent claim 35, from which claims 49-51 depend, is not obvious over Petersen and Hung et al. Applicant submits that even if one used the disclosure of Isner et al. to treat disorders of the heart, it would not have been obvious to one skilled in the art to use SDF-1 α in the claimed method which is not obvious over Petersen when combined with Hung et al. and Rempel et al.

Applicant's arguments have been fully considered but are not found to be persuasive. The Examiner's response to Applicant's arguments against Petersen and Hung et al. are

presented in sections 2(i)-(iii), above. As discussed in the previous Office Action, Isner et al. teaches a method for increasing vascularization comprising administering to a mammal an effective amount of a vascularization modulating agent, such as stromal derived factor-1 (SDF-1) (bottom of page 4 through the top of page 5). Isner et al. disclose that the methods of the invention have a wide spectrum of uses in a human patient, i.e., use in the prevention or treatment of at least cerebrovascular ischemia, ischemic cardiopathy, and myocardial ischemia (page 15, lines 1-5). Isner et al. also teach that the ischemia may be especially adversely impact hear or brain tissue as often occurs in cardiovascular disease or stroke (page 15, lines 6-10). Isner et al. indicate that agents can be administered directly, i.e., intra-arterially (page 19, lines 17-18).

Hence, it would have been obvious to the person of ordinary skill in the art at the time the invention was made to modify the method of intramyocardially or intracoronarily administering SDF-1 α to heart tissue as taught by Peterson and Hung et al. to treat disorders of heart tissue as taught by Isner et al. The person of ordinary skill in the art would have been motivated to make that modification in order to localize cell migration/differentiation and tissue repair to the ischemic heart (see for example, Hung et al. page 1, [0007]; Petersen, page 1 [0004, 0006]). The person of ordinary skill in the art reasonably would have expected success because similar proteins and agents were already being intramyocardially and intracoronarily administered to the heart to treat ischemic diseases at the time the invention was made (see Hung et al.).

Conclusion

No claims are allowable.

THIS ACTION IS MADE FINAL. Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire **THREE MONTHS** from the mailing date of this action. In the event a first reply is filed within **TWO MONTHS** of the mailing date of this final action and the advisory action is not mailed until after the end of the **THREE-MONTH** shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than **SIX MONTHS** from the mailing date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Bridget E. Bunner whose telephone number is (571) 272-0881. The examiner can normally be reached on 9:00-5:30 M-F.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Gary Nickol can be reached on (571) 272-0835. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

BEB
Art Unit 1647
05 January 2011

/Bridget E Bunner/
Primary Examiner, Art Unit 1647